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NEWS	2	OCT 04	Precision of EMBASE searching enhanced with new chemical name field
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NEWS	4	OCT 21	CA/CAPLUS kind code changes for Chinese patents increase consistency, save time
NEWS	5	OCT 22	New version of STN Viewer preserves custom highlighting of terms when patent documents are saved in .rtf format
NEWS	6	OCT 28	INPADOCDB/INPAFAMDB: Enhancements to the US national patent classification.
NEWS	7	NOV 03	New format for Korean patent application numbers in CA/CAPLUS increases consistency, saves time.
NEWS	8	NOV 04	Selected STN databases scheduled for removal on December 31, 2010
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NEWS	10	NOV 22	Higher System Limits Increase the Power of STN Substance-Based Searching
NEWS	11	NOV 24	Search an additional 46,850 records with MEDLINE backfile extension to 1946
NEWS	12	DEC 14	New PNK Field Allows More Precise Crossover among STN Patent Databases
NEWS	13	DEC 18	ReaxysFile available on STN
NEWS	14	DEC 21	CAS Learning Solutions -- a new online training experience
NEWS	15	DEC 22	Value-Added Indexing Improves Access to World Traditional Medicine Patents in CAPLUS
NEWS	16	JAN 24	The new and enhanced DPCI file on STN has been released
NEWS	17	JAN 26	Improved Timeliness of CAS Indexing Adds Value to USPATFULL and USPAT2 Chemistry Patents
NEWS	18	JAN 26	Updated MeSH vocabulary, new structured abstracts, and other enhancements improve searching in STN reload of MEDLINE
NEWS	19	JAN 28	CABA will be updated weekly
NEWS	20	FEB 23	PCTFULL file on STN completely reloaded
NEWS	21	FEB 23	STN AnaVist Test Projects Now Available for Qualified Customers
NEWS	22	FEB 25	LPCI will be replaced by LDPCI
NEWS	23	MAR 07	Pricing for SELECTing Patent, Application, and Priority Numbers in the USPAT and IFI Database Families is Now Consistent with Similar Patent Databases on STN
NEWS	24	APR 26	Expanded Swedish Patent Application Coverage in CA/CAPLUS Provides More Current and Complete Information
NEWS	25	APR 28	The DWPI (files WPINDEX, WPIDS and WPIX) on STN have been enhanced with thesauri for the European Patent Classifications

NEWS 26 MAY 02 MEDLINE Improvements Provide Fast and Simple Access to DOI and Chemical Name Information

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DICTIONARY FILE UPDATES: 8 MAY 2011 HIGHEST RN 1291818-64-8

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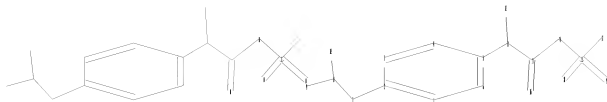
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chain nodes :
7 8 9 10 11 12 13 14 15 16 17 18 19
ring nodes :
1 2 3 4 5 6
chain bonds :
2-7 5-11 7-8 8-9 8-10 11-12 11-13 13-14 13-16 14-15 15-17 15-18 15-19
ring bonds :
1-2 1-6 2-3 3-4 4-5 5-6
exact/norm bonds :
13-14 13-16 14-15 15-17 15-18 15-19
exact bonds :
2-7 5-11 7-8 8-9 8-10 11-12 11-13
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6

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Match level :
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11:CLASS 12:CLASS 13:CLASS 14:CLASS 15:CLASS 16:CLASS 17:CLASS 18:CLASS
19:CLASS

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L1 STRUCTURE UPLOADED

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FULL SEARCH INITIATED 11:38:15 FILE 'REGISTRY'
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SEARCH TIME: 00.00.01

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FULL ESTIMATED COST	77.76	77.99

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FILE COVERS 1907 - 9 May 2011 VOL 154 ISS 20
FILE LAST UPDATED: 8 May 2011 (20110508/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2011
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2011

CAPLUS now includes complete International Patent Classification (IPC) reclassification data for the fourth quarter of 2010.

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=> s l2
L3 33 L2

=> s l3 not py>2005
8200802 PY>2005
L4 9 L3 NOT PY>2005

=> d l4 ibib abs 1-9

L4 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2011 ACS on STN
ACCESSION NUMBER: 2005:1301378 CAPLUS
DOCUMENT NUMBER: 144:324102
TITLE: Neutrophil recruitment in the reperfused-injured rat liver was effectively attenuated by repertaxin, a novel allosteric non-competitive inhibitor of CXCL8 receptors: A therapeutic approach for the treatment of post-ischemic hepatic syndromes
AUTHOR(S): Cavalieri, B.; Mosca, M.; Ramadori, P.; Perrelli, M.-G.; De Simone, L.; Colotta, F.; Bertini, R.; Poli, G.; Cutrin, J. C.
CORPORATE SOURCE: Laboratory of Experimental Liver Pathology, Department of Clinical and Biological Sciences, University of

SOURCE: Turin, L'Aquila, Italy
 International Journal of Immunopathology and
 Pharmacology (2005), 18(3), 475-486
 CODEN: IJIFE4; ISSN: 0394-6320
 PUBLISHER: Biolife s.a.s.
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Hepatic reperfusion injury represents a crucial problem in several clin.
 situations including liver transplantation, extensive hepatectomy and
 hypovolemic shock with resuscitation. Repertaxin is a new non-competitive
 allosteric blocker of interleukin-8 (CXCL8) receptors, which by locking
 CXCR1/R2 in an inactive conformation, prevents receptor signaling and
 polymorphonuclear leukocyte (PMN) chemotaxis. The present study shows
 that repertaxin dramatically prevents rat post-ischemic hepatocellular
 necrosis (80% of inhibition) and PMN infiltration (96% of inhibition) at a
 clin.-relevant time (24 h) of reperfusion. Treatment with repertaxin by
 continuous infusion is demonstrated to be the optimal route of
 administration of the compound especially in view of its clin. therapeutic use.
 Because repertaxin has proven to be safe and well tolerated in different
 animal studies and in phase I studies in human volunteers, it is in fact a
 candidate novel therapeutic agent for the prevention and treatment of
 hepatic post-ischemic injury.
 OS.CITING REF COUNT: 17 THERE ARE 17 CAPLUS RECORDS THAT CITE THIS
 RECORD (17 CITINGS)
 REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS
 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
 L4 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2011 ACS on STN
 ACCESSION NUMBER: 2005:460353 CAPLUS
 DOCUMENT NUMBER: 143:145782
 TITLE: 2-Arylpropionic CXC Chemokine Receptor 1 (CXCR1)
 Ligands as Novel Noncompetitive CXCL8 Inhibitors
 AUTHOR(S): Allegretti, Marcello; Bertini, Riccardo; Cesta, Maria
 Candida; Bizzarri, Cinzia; Di Bitondo, Rosa; Di
 Cioccio, Vito; Galliera, Emanuela; Berdini, Valerio;
 Topai, Alessandra; Zampella, Giuseppe; Russo,
 Vincenzo; Di Bello, Nicoletta; Nano, Giuseppe;
 Nicolini, Luca; Locati, Massimo; Fantucci, Piercarlo;
 Florio, Saverio; Colotta, Francesco
 CORPORATE SOURCE: Dompe Research and Development, Dompe S.p.A.,
 L'Aquila, 67100, Italy
 SOURCE: Journal of Medicinal Chemistry (2005), 48(13),
 4312-4331
 CODEN: JMCMAR; ISSN: 0022-2623
 PUBLISHER: American Chemical Society
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 OTHER SOURCE(S): CASREACT 143:145782
 AB The CXC chemokine CXCL8/IL-8 plays a major role in the activation and
 recruitment of polymorphonuclear (PMN) cells at inflammatory sites. CXCL8
 activates PMNs by binding the seven-transmembrane (7-TM) G-protein-coupled
 receptors CXC chemokine receptor 1 (CXCR1) and CXC chemokine receptor 2
 (CXCR2). (R)-Ketoprofen (1) was previously reported to be a potent and
 specific noncompetitive inhibitor of CXCL8-induced human PMNs chemotaxis.
 The authors report here mol. modeling studies showing a putative
 interaction site of 1 in the TM region of CXCR1. The binding model was
 confirmed by alanine scanning mutagenesis and photoaffinity labeling
 expts. The mol. model driven medicinal chemical optimization of 1 led to a
 new class of potent and specific inhibitors of CXCL8 biol. activity.
 Among these, repertaxin was selected as a clin. candidate drug for
 prevention of postischemia reperfusion injury.
 OS.CITING REF COUNT: 40 THERE ARE 40 CAPLUS RECORDS THAT CITE THIS

REFERENCE COUNT: 59 RECORD (41 CITINGS)
THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2005:437986 CAPLUS

DOCUMENT NUMBER: 143:53210

TITLE: Inhibition of the chemokine receptor CXCR2 prevents
kidney graft function deterioration due to
ischemia/reperfusion

AUTHOR(S): Cugini, Daniela; Azzollini, Nadia; Gagliardini, Elena;
Cassisi, Paola; Bertini, Riccardo; Colotta, Francesco;
Noris, Marina; Remuzzi, Giuseppe; Benigni, Ariela

CORPORATE SOURCE: Transplant Research Center "Chiara Cucchi de
Alessandri e Gilberto Crespi" Mario Negri Institute
for Pharmacological Research, Bergamo, Italy

SOURCE: Kidney International (2005), 67(5), 1753-1761

CODEN: KDYIA5; ISSN: 0085-2538

PUBLISHER: Blackwell Publishing, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Background: Ischemia/reperfusion (I/R) injury after organ transplantation is a major cause of delayed graft function. Following I/R, locally produced CXC chemokines attract and activate granulocytes, which in turn promote graft damage. Methods: We examined the involvement of granulocyte recruitment via the CXCR2 pathway in a rat model of 4 h cold ischemia followed by kidney transplantation. Serum creatinine and intragraft granulocyte infiltration were monitored in the early phase posttransplant. A CXCR2 inhibitor, repertaxin, was given to recipients before transplantation (at -24 h or -8 h or -2 h), immediately before reperfusion and 2 h later. Results: An increase of granulocyte chemoattractant CINC-1/interleukin-8 (IL-8) mRNA expression after I/R both in syngeneic and allogeneic transplantation was associated with a marked infiltration of granulocytes in renal tissue. In syngeneic transplantation, Lewis rats given 15 mg/kg repertaxin 24 h before surgery had granulocyte graft infiltration and serum creatinine levels significantly reduced in respect to vehicle-treated animals. Intermediate effects were observed with 5 mg/kg, whereas the dose of 30 mg/kg had toxic effects. We found that reducing the pretreatment time to 8 h before surgery was still effective. Prevention of granulocyte infiltration and serum creatinine increase was also obtained in allogeneic transplantation, when Brown Norway recipients of Lewis kidneys were given 15 mg/kg repertaxin starting 8 h before surgery. Conclusion: Repertaxin treatment of the recipient animal was effective in preventing granulocyte infiltration and renal function impairment both in syngeneic and in allogeneic settings. The possibility to modulate I/R injury in this rat model opens new perspectives for preventing posttransplant delayed graft function in humans.

OS.CITING REF COUNT: 46 THERE ARE 46 CAPLUS RECORDS THAT CITE THIS
RECORD (46 CITINGS)

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2005:319144 CAPLUS

DOCUMENT NUMBER: 142:475974

TITLE: Neuroprotection with the CXCL8 inhibitor repertaxin in
transient brain ischemia

AUTHOR(S): Garau, Angela; Bertini, Riccardo; Colotta, Francesco;
Casilli, Federica; Bigini, Paolo; Cagnotto, Alfredo;
Mennini, Tiziana; Ghezzi, Pietro; Villa, Pia

CORPORATE SOURCE: "Mario Negri" Institute for Pharmacological Research,
Milan, Italy

SOURCE: Cytokine+ (2005), 30(3), 125-131
CODEN: CYTIE9; ISSN: 1043-4666
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Infiltration of polymorphonuclear neutrophils (PMNs) is thought to play a role in ischemic brain damage. The present study investigated the effect of repertaxin, a new noncompetitive allosteric inhibitor for the receptors of the inflammatory chemokine CXCL8 (CXCL8)/interleukin-8 (IL-8), on PMN infiltration and tissue injury in rats. Cerebral ischemia was induced by permanent or transient occlusion of the middle cerebral artery and myeloperoxidase activity, a marker of PMN infiltration, and infarct volume were evaluated 24 h later. Repertaxin (15 mg/kg) was administered systemically at the time of ischemia and every 2 h for four times. In permanent ischemia repertaxin reduced PMN infiltration by 40% in the brain cortex but did not limit tissue damage. In transient ischemia (90-min ischemia followed by reperfusion), repertaxin inhibited PMN infiltration by 54% and gave 44% protection from tissue damage. Repertaxin had anti-inflammatory and neuroprotective effects also when given at reperfusion and even at 2 h of reperfusion. The protective effect of repertaxin did not interfere with brain levels of the chemokine. Since the PMN infiltration and its inhibition by repertaxin were comparable in the two models we conclude that reperfusion induces PMN activation, and inhibition of CXCL8 by repertaxin might be of pharmacol. interest in transient ischemia.

OS.CITING REF COUNT: 29 THERE ARE 29 CAPLUS RECORDS THAT CITE THIS RECORD (29 CITINGS)
REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2011 ACS ON STN

ACCESSION NUMBER: 2005:201863 CAPLUS
DOCUMENT NUMBER: 142:385080
TITLE: Predicting Human Serum Albumin Affinity of Interleukin-8 (CXCL8) Inhibitors by 3D-QSPR Approach
AUTHOR(S): Aureli, Loretta; Cruciani, Gabriele; Cesta, Maria Candida; Anacardio, Roberto; De Simone, Lucio; Moriconi, Alessio
CORPORATE SOURCE: Molecular Discovery Ltd., London, W1A 3BQ, UK
SOURCE: Journal of Medicinal Chemistry (2005), 48(7), 2469-2479
CODEN: JMCMAR; ISSN: 0022-2623
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 142:385080

AB A novel class of 2-(R)-phenylpropionamides has been recently reported to inhibit in vitro and in vivo interleukin-8 (CXCL8)-induced biol. activities. These CXCL8 inhibitors are derivs. of phenylpropionic nonsteroidal antiinflammatory drugs (NSAIDs), high-affinity ligands for site II of human serum albumin (HSA). Up to date, only a limited number of in silico models for the prediction of albumin protein binding are available. A three-dimensional quant. structure-property relationship (3D-QSPR) approach was used to model the exptl. affinity constant (K_i) to plasma proteins of 37 structurally related molcs., using physicochem. and 3D-pharmacophoric descriptors. Mol. docking studies highlighted that training set molcs. preferentially bind site II of HSA. The obtained model shows satisfactory statistical parameters both in fitting and predicting validation. External validation confirmed the statistical significance of the chemometric model, which is a powerful tool for the prediction of HSA binding in virtual libraries of structurally related compds.

OS.CITING REF COUNT: 16 THERE ARE 16 CAPLUS RECORDS THAT CITE THIS

REFERENCE COUNT: 47 RECORD (16 CITINGS)
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RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2005:28032 CAPLUS

DOCUMENT NUMBER: 142:190637

TITLE: Inhibition of interleukin-8 (CXCL8/IL-8) responses by
repertaxin, a new inhibitor of the chemokine receptors
CXCR1 and CXCR2

AUTHOR(S): Casilli, Federica; Bianchini, Andrea; Gloaguen,
Isabelle; Biordi, Leda; Allesse, Edoardo; Festuccia,
Claudio; Cavalieri, Barbara; Strippoli, Raffaele;
Cervellera, Maria Neve; Di Bitondo, Rosa; Ferretti,
Elisabetta; Mainiero, Fabrizio; Bizzarri, Cinzia;
Colotta, Francesco; Bertini, Riccardo

CORPORATE SOURCE: Dompe S.p.A. Research Center, L'Aquila, Italy

SOURCE: Biochemical Pharmacology (2005), 69(3), 385-394

CODEN: BCPA6; ISSN: 0006-2952

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Repertaxin is a new non-competitive allosteric blocker of interleukin-8
(CXCL8/IL-8) receptors (CXCR1/R2), which by locking CXCR1/R2 in an
inactive conformation prevents receptor signaling and human
polymorphonuclear leukocyte (PMN) chemotaxis. Given the unique mode of
action of repertaxin it was important to examine the ability of repertaxin
to inhibit a wide range of biol. activities induced by CXCL8 in human
leukocytes. Our results show that repertaxin potently and selectively
blocked PMN adhesion to fibrinogen and CD11b up-regulation induced by
CXCL8. Reduction of CXCL8-mediated PMN adhesion by repertaxin was paralleled
by inhibition of PMN activation including secondary and tertiary granule
release and pro-inflammatory cytokine production, whereas PMN phagocytosis of
Escherichia coli bacteria was unaffected. Repertaxin also selectively
blocked CXCL8-induced T lymphocyte and natural killer (NK) cell migration.
These data suggest that repertaxin is a potent and specific inhibitor of a
wide range of CXCL8-mediated activities related to leukocyte recruitment
and functional activation in inflammatory sites.

OS.CITING REF COUNT: 31 THERE ARE 31 CAPLUS RECORDS THAT CITE THIS
RECORD (31 CITINGS)

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2004:803495 CAPLUS

DOCUMENT NUMBER: 141:343217

TITLE: Repertaxin, a novel inhibitor of rat CXCR2 function,
inhibits inflammatory responses that follow intestinal
ischaemia and reperfusion injury

AUTHOR(S): Souza, Danielle G.; Bertini, Riccardo; Vieira,
Angelica T.; Cunha, Fernando Q.; Poole, Steve;
Allegretti, Marcello; Colotta, Francesco; Teixeira,
Mauro M.

CORPORATE SOURCE: Immunopharmacology, Departamento de Bioquímica e
Imunologia, ICB, Universidade Federal de Minas Gerais,
Belo Horizonte, Brazil

SOURCE: British Journal of Pharmacology (2004), 143(1),
132-142

CODEN: BJPCBM; ISSN: 0007-1188

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Neutrophils are thought to play a major role in the mediation of reperfusion injury. CXC chemokines are known inducers of neutrophil recruitment. Here, we assessed the effects of Repertaxin, a novel low mol. weight inhibitor of human CXCL8 receptor activation, on the local, remote and systemic injuries following intestinal ischemia and reperfusion (I/R) in the rat. Pre-incubation of rat neutrophils with Repertaxin (10-11-10-6 M) inhibited the chemotaxis of neutrophils induced by human CXCL8 or rat CINC-1, but not that induced by fMLP, PAF or LTB4, in a concentration-dependent manner. Repertaxin also prevented CXCL8-induced calcium

influx but not CXCL8 binding to purified rat neutrophils. In a model of mild I/R injury (30 min of ischemia and 30 min of reperfusion), Repertaxin dose-dependently (3-30 mg kg-1) inhibited the increase in vascular permeability and neutrophil influx. Maximal inhibition occurred at 30 mg kg-1. Following severe I/R injury (120 min of ischemia and 120 min of reperfusion), Repertaxin (30 mg kg-1) markedly prevented neutrophil influx, the increase in vascular permeability both in the intestine and the lungs. Moreover, there was prevention of hemorrhage in the intestine of reperfused animals. Repertaxin effectively suppressed the increase in tissue (intestine and lungs) and serum concns. of TNF- α and the reperfusion-associated lethality. For comparison, we also evaluated the effects of an anti-CINC-1 antibody in the model of severe I/R injury. Overall, the antibody effectively prevented tissue injury, systemic inflammation and lethality. However, the effects of the antibody were in general of lower magnitude than those of Repertaxin. In conclusion, CINC-1 and possibly other CXC chemokines, acting on CXCR2, have an important role during I/R injury. Thus, drugs, such as Repertaxin, developed to block the function of the CXCR2 receptor may be effective at preventing reperfusion injury in relevant clin. situations.

OS.CITING REF COUNT: 51 THERE ARE 51 CAPLUS RECORDS THAT CITE THIS RECORD (51 CITINGS)
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2004:703810 CAPLUS

DOCUMENT NUMBER: 141:343408

TITLE: Noncompetitive allosteric inhibitors of the inflammatory chemokine receptors CXCR1 and CXCR2: Prevention of reperfusion injury

AUTHOR(S): Bertini, Riccardo; Allegretti, Marcello; Bizzarri, Cinzia; Moriconi, Alessio; Locati, Massimo; Zampella, Giuseppe; Cervellera, Maria N.; di Ciccio, Vito; Cesta, Maria C.; Galliera, Emanuela; Martinez, Fernando O.; di Bitondo, Rosa; Trolani, Giulia; Sabbatini, Vilma; D'Anniballe, Gaetano; Anacardio, Roberto; Cutrin, Juan C.; Cavallieri, Barbara; Mainiero, Fabrizio; Strippoli, Raffaele; Villa, Pia; di Girolamo, Maria; Martin, Franck; Gentile, Marco; Santoni, Angela; Corda, Daniela; Poli, Giuseppe; Mantovani, Alberto; Ghezzi, Pietro; Colotta, Francesco

CORPORATE SOURCE: Dompe, L'Aquila, 67100, Italy
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2004), 101(32), 11791-11796
CODEN: PNASA6; ISSN: 0027-8424

PUBLISHER: National Academy of Sciences

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The chemokine CXC ligand 8 (CXCL8)/IL-8 and related agonists recruit and activate polymorphonuclear cells by binding the CXC chemokine receptor 1 (CXCR1) and CXCR2. Here the authors characterize the unique mode of action of a small-mol. inhibitor (repertaxin) of CXCR1 and CXCR2.

Structural and biochem. data are consistent with a noncompetitive allosteric mode of interaction between CXCR1 and repertaxin, which, by locking CXCR1 in an inactive conformation, prevents signaling. Repertaxin is an effective inhibitor of polymorphonuclear cell recruitment in vivo and protects organs against reperfusion injury. Targeting the repertaxin interaction site of CXCR1 represents a general strategy to modulate the activity of chemoattractant receptors.

OS.CITING REF COUNT: 106 THERE ARE 106 CAPLUS RECORDS THAT CITE THIS RECORD (106 CITINGS)
 REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2004:498365 CAPLUS

DOCUMENT NUMBER: 141:173953

TITLE: Acylmethanesulfonamides as new acylating agents for primary amines

AUTHOR(S): Coniglio, Silvia; Aramini, Andrea; Cesta, M. Candida; Colagioia, Sandro; Curti, Roberto; D'Alessandro, Fabrizio; D'Anniballe, Gaetano; D'Elia, Valerio; Nano, Giuseppe; Orlando, Valerie; Allegretti, Marcello

CORPORATE SOURCE: Dompe Research and Development, Chemistry Department, Dompe S.p.A., L'Aquila, 67100, Italy

SOURCE: Tetrahedron Letters (2004), 45(28), 5375-5378

CODEN: TELEAY; ISSN: 0040-4039

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

OTHER SOURCE(S): CASREACT 141:173953

AB A simple and efficient procedure for the preparation of secondary amides through internal condensation of acylmethanesulfonamides ammonium salts is described. The selective acylation of mixed primary-secondary amines could be an attractive application of this method.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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SINCE FILE	TOTAL
ENTRY	SESSION
32.20	114.09

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
-7.83	-7.83

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